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EXOGENOUS Fe^{+2} FAILS TO ENHANCE ETHANOL-INDUCED LIPID PEROXIDATION IN DEVELOPING CHICK BRAINS. Daniel J. Coughlin and Dr. Robert R. Miller, Jr.*, Hillsdale College, Biology Department, 33 E. College St., Hillsdale, MI 49242, email: bob.miller@hillsdale.edu*.

Embryonic ethanol (EtOH) exposure is known to reduce the levels of polyunsaturated long-chain membrane fatty acids and increase the levels of saturated short-chain membrane fatty acids. These EtOH-induced decreases in long-chain / short membrane fatty acids and unsaturated / saturated membrane fatty acids correlate with EtOH-induced reductions in brain neuron densities within the cerebral hemispheres and optic lobes and increased levels of brain lipid hydroperoxides. Recent work has demonstrated that exogenous treatments of either α-tocopherol or γ-tocopherol attenuate EtOH-induced changes in membrane fatty acid composition and EtOH-induced changes in brain morphology. Thus, EtOH-impaired brain development appears to be associated with EtOH-induced lipid peroxidation.

Because oxygen radicals and hydroxyl radicals can be generated through the Fenton reaction, we asked whether or not exogenous Fe^{+2} (3.003 μmol $FeCl_2$ / kg egg) accentuated EtOH-induced (3.025 mmol / kg egg) changes in brain membrane lipid composition. While EtOH-induced and Fe^{+2}-induced reductions in the ratios of long-chain / short-chain membrane fatty acids and the ratios of unsaturated / saturated membrane fatty acids were observed, exogenous Fe^{+2} failed to enhance EtOH-induced reductions in long-chain polyunsaturated membrane fatty acids. Hence, a compensatory mechanism(s) exists which protects against membrane lipid peroxidation in developing chick embryos.